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HEMORRHAGIC MENINGOENCEPHALITIS IN ANTHRAX

A REPORT OF THREE CASES

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Although there are reasons to believe that localization of the bacilli in the brain is relatively common[1] in anthrax the cerebral changes produced are, so far as I am able to find, as yet undescribed in American literature. As there is at present an increasing incidence of anthrax in this country, a report at this time I believe is appropriate, especially in so far as it may assist in the recognition of such infections either clinically or at the necropsy table.

Three of the deaths from anthrax in Chicago within the past 6 months have yielded the brains on which this report is based and in only one of these was anthrax suspected before death. These 3 deaths were subject to medicolegal inquiry.

Case 1.—The first case was that of a white boy, aged 15 years, who was admitted, in coma, to the Cook County Hospital, service of Dr. Friedberg. From the father a history was obtained of onset two days before with headache and sore throat and also the history of an old ear trouble. On admission the temperature was 99.4 F., pulse 120, respirations 26; physical examination revealed an acutely ill young man in coma with a swelling of the left side of the neck, hyperemia and edema of the vocal cords, and a leukocyte count of 75,000 (97% polymorphonuclears). He lived 4 hours in the hospital and died 65 hours after the onset. A clinical diagnosis was not made, but mastoid abscess with lateral sinus thrombosis was suspected.

Anatomic diagnosis (Dr. E. R LeCount): Acute hemorrhagic leptomeningoencephalitis (anthrax infection); anthrax of the left side of the neck, scalp and chest; hyperemic papules of the face (anthrax?); acute anemia.

The skull cap came away with ease. The dura was everywhere tense and was light purple from the blood underneath. In the superior longitudinal sinus there was fluid blood only. The brain was everywhere on top bright red from the blood in the pia arachnoid meshwork. There was a little more blood in the front than in the back half; there was enough in the front half to hide entirely the convolutions, while in the back half there was only a thin layer of blood on the summits of the convolutions, the sulci here being filled. There was a small amount of viscid blood stained material between the two layers of the arachnoid but no free blood. Where the pia arachnoid was stripped from the surface of the convolutions numerous red pinpoint spots remained, as large as 1 mm. in diameter, which stippled these surfaces quite uniformly. The brain substance was so swollen that the convolutions were

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[1] Lancet, 1880, 1, pp. 871 and 909; 1901, 2, p. 133; 1905, 2, p. 934.

almost flat on their tops, and the sulci almost closed. The brain, especially the gray matter, was soft. The large arteries of the base of the brain were unchanged. The cerebrospinal fluid was blood stained. The brain was put in formalin solution and the examination continued three days later.

On the two broad surfaces made by one horizontal cut through the corona radiata there were hundreds of 1 to 2 mm. petechial hemorrhages, mostly oblong and irregular, limited practically to the cortex, and as a rule perpendicular to the outside surface of the brain (fig. 1,a). They were about the same distance apart and had a tendency to widen out in the deeper layers of the cortex (fig. 1,b). There were few such hemorrhages in the white matter, but the small vessels here were greatly distended with blood. A similar section was made through the basal ganglions (fig. 1), and in addition to the characteristically distributed cortical hemorrhages there were numerous hemorrhages in the lenticular and caudate nuclei, both thalami, and the hippocampi. These hemorrhages were not confluent as they may seem in the photograph, but were separated by narrow bands of dark gray brain substance. The largest of these places of hemorrhage had a surface measurement of 1.5 by 5 mm. The hemorrhages of the right thalamus and head of the right caudate nucleus were so numerous as to cause these structures to bulge into and narrow the adjacent ventricles (fig. 1). There were three or four rather large (1 to 3 mm.) hemorrhages of the gray matter of the left dentate nucleus of the cerebellum.

Bacteriology.—Anthrax was not suspected until after death. Cultures were made from the brain substance, superior longitudinal sinus blood, and cerebrospinal fluid and from each a gram-positive rod shaped bacillus grew in pure culture on human blood-agar plates. This bacillus answered all of the cultural and morphologic requirements for anthrax and killed guinea-pigs inoculated subcutaneously in 12 to 18 hours, producing anthrax symptoms, large spleens, and anthracemia (encapsulated bacilli if examined before the death of the pig). The bacillus was also grown in pure culture from the fluids and tissues of these pigs. Meningeal hemorrhage was not found in either of the two pigs examined, but it is known to occur from such experiments (Gilmour and Campbell[2]).

Microscopic Examination.—A perfect series of sections was made from the cerebral cortex at a place chosen at random and cut tangent to the outside surface, beginning at the visceral layer of the arachnoid, extending through the cortex and into the white matter for 6 to 7 mm. Sections not in series were taken from other places and corresponded in all details to those of the series.

The visceral layer of the arachnoid was in places two or three times its normal thickness due to the separation of its delicate connective tissue fibers by a cellular, slightly fibrinous and bacillus laden exudate, similar to that in the pia arachnoid meshwork. Within the meshes of the pia arachnoid there was a large amount of exudate and great numbers of bacilli, in the spaces mostly red blood cells. This exudate was for the most part composed of red blood cells and leukocytes (large mononuclear and polymorphonuclear). There were also a few endothelial and plasma cells, large vacuolated mononuclear cells, and a small amount of fibrin. Intermixed with this there were enormous numbers of bacilli. Nowhere was the exudate entirely free from red blood cells, but the latter were most numerous in the dilated spaces. There was very little disintegration of the leukocytes of the exudate, a few of which, however, contained whole or parts of broken bacilli.

Canadian Med. Assn. Jour., 1918, 8, p. 97.

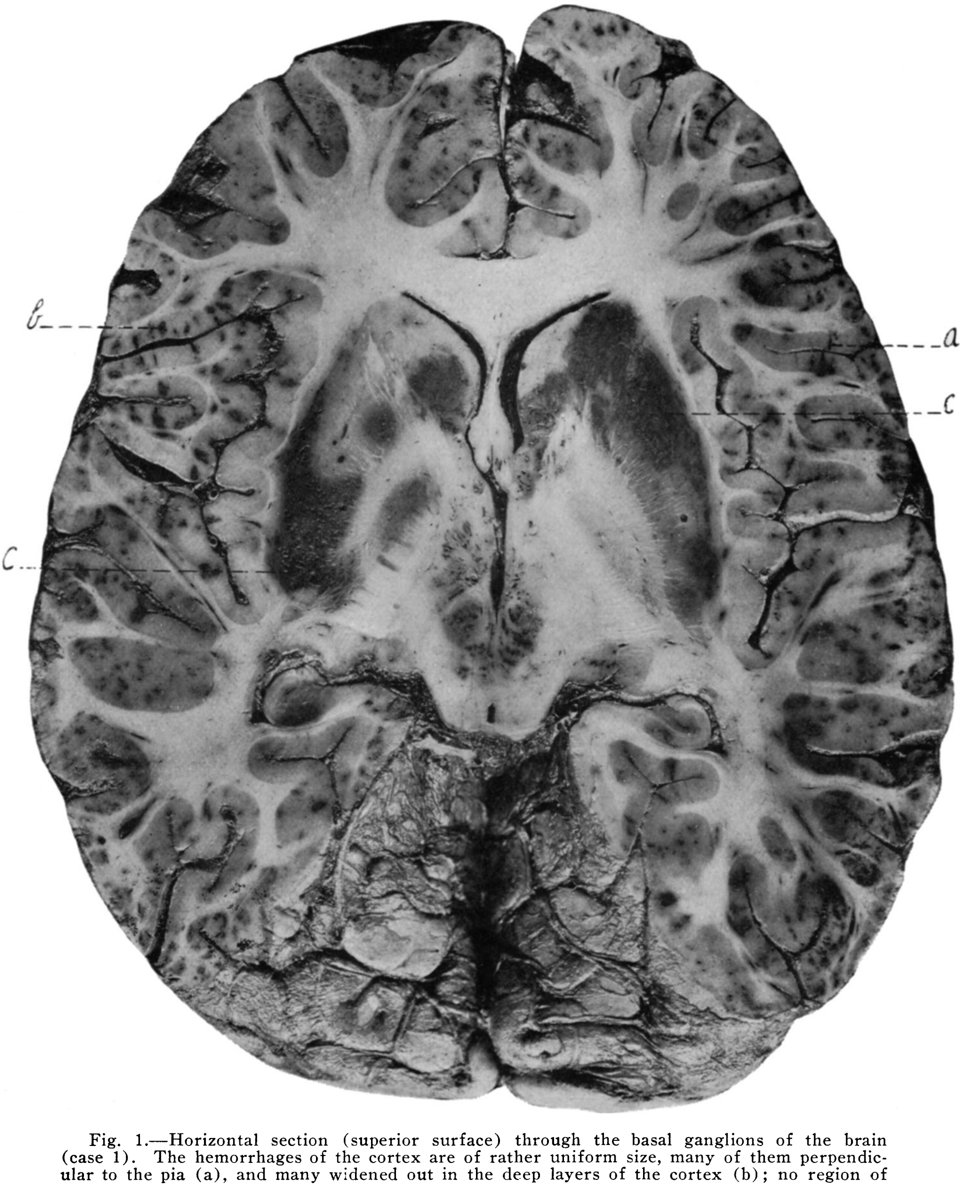

Fig. 1.—Horizontal section (superior surface) through the basal ganglions of the brain (case 1). The hemorrhages of the cortex are of rather uniform size, many of them perpendicular to the pia (a), and many widened out in the deep layers of the cortex (b); no region of the cortex is spared; very few hemorrhages of the white matter; the hemorrhages in the lenticular and caudate nuclei are small, not confluent, but separated by dark gray strands of brain tissue. Note the predominance of hemorrhages (c) along the course of Charcot's artery in the outer portion of each lenticular nucleus.

The chief changes in the leptomeninges were those of the vessel walls. The adventitia of practically every vessel (artery and vein) was laden with bacilli and leukocytes. The walls of these vessels were thicker than normal, the individual elements being spread wide apart by edema. As a result the walls were weakened and it was common to find them ruptured. The intimal changes were least marked and consisted of patchy proliferation of the endothelium and subendothelial infiltration with leukocytes (fig. 6). It was on such places as this that thrombi were found. There was an increased number of leukocytes in the lumens of all the cerebral vessels.

One of the early changes of the wall was a partial or total separation of the adventitia from the media that may be present with a still intact media or after the rupture of the media with a still intact intima, in the last instance forming what is described as "aneurysma desiccans." In a few arteries the wall was greatly infiltrated with red blood cells and leukocytes, the intima being still intact, bridging over the defect. This was not so common in the veins.

The delicate fibers of the pia mater were moderately separated by exudate and bacilli. Bacilli were also numerous in the adventitia sheaths of the arteries and veins as they entered the cortex and could be traced in this situation through the entire gray matter and into the underlying white substance. Mixed with these bacilli was a hemorrhagic leukocytic exudate which could be traced back to that of the pia mater (as in fig. 2 b,c). Between the pia and the surface of the brain substance there was, in places, a layer of finely granular coagulated plasma as thick as 300 mikrons, with an occasional nonstaining cell. In this layer of plasma and below it there were occasional bacilli. Between the pia and the brain there were also, in places, numerous red blood cells which were often in direct continuity with similar cells in the perivascular spaces of the brain substance (fig. 2 d, d′).

In the gray matter of the cerebrum the essential changes were vascular. Hardly a single arteriole or venule was unchanged. There were bacilli in the adventitia of these vessels. In the perivascular space surrounding practically every vessel there were red blood cells, so numerous in some as to distend them to a diameter 12 or 15 times greater than the vessel. If such a vessel were followed along in the serial sections, one or more places of rupture might be found in its wall. From these dilated perivascular spaces the red blood cells extended out in irregular finger-like masses, forming the petechial hemorrhages seen grossly. In these hemorrhages, as well as in the perivascular spaces, there were only a few scattered bacilli except in the neighborhood (100 to 200 mikrons) of the breaks in the walls of the vessels. Here the bacilli, present only in the adventitial spaces at other levels, occurred in relatively small numbers, both in the perivascular spaces, in the places of hemorrhage, and even in the surrounding brain substance. The venules of the gray matter were altered to a slightly greater degree than the arterioles.

As the white matter of the cerebrum was approached in a study of the serial sections, all these changes became progressively less. The changes of edema of the interstitial brain substance, the granular changes in the protoplasm of the ganglion cells, became less. The loss of chromatin and the poor staining qualities (small darkly stained nucleus or faintly staining nucleus) of the nuclei of the ganglion cells was less marked in the deeper parts of the gray matter. In the white matter the lumens of the vessels were greatly distended with red blood cells. In no section were bacilli found inside the vessel lumens, either of the brain or its meninges.

CASE 2.—A. K., a laborer, aged 62 years, entered Cook Coutny Hospital, the service of Dr. Hollinger, in coma, suffering from air hunger and cyanosis and with an indefinite history of an onset three days previously. He was a laborer in a "curled hair" establishment handling hair from South America.

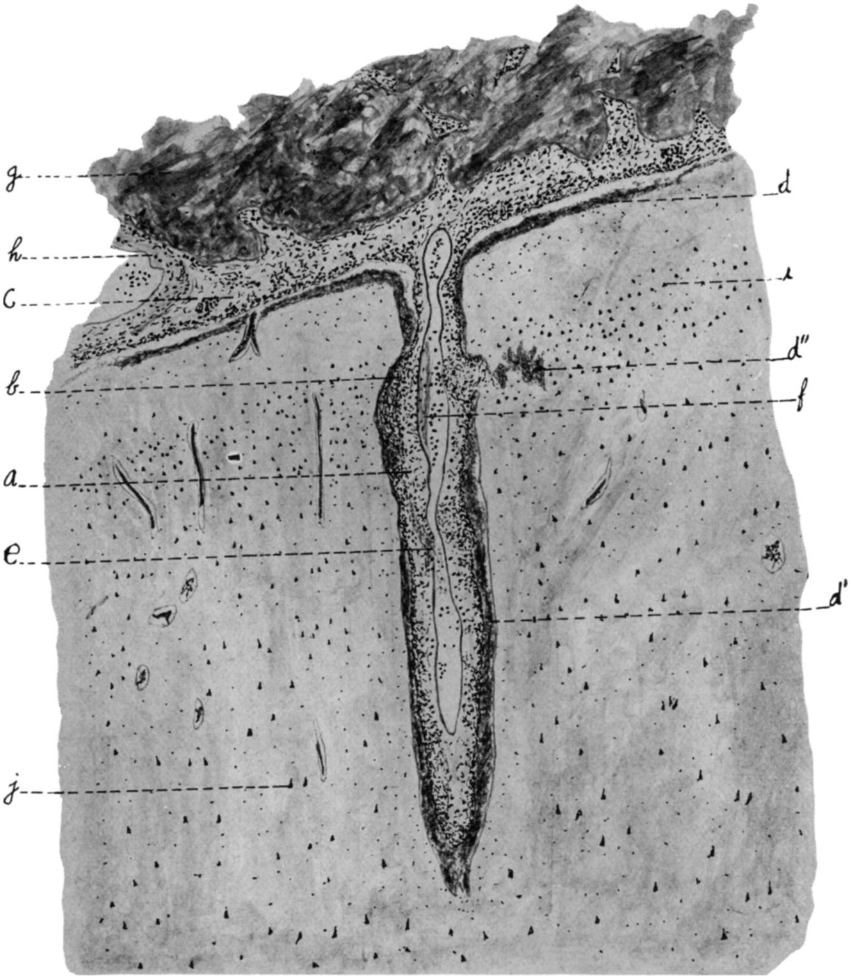

Fig. 2.—Longitudinal section of an arteriole entering horizontally the substance of the left middle frontal gyrus from the superior frontal sulcus (case 2). (A similar vessel is shown cut cross-wise in fig. 3.) The fibers of the adventitia (a) are dissected widely apart by edema and in this layer of the vessel there are numerous leukocytes mixed with bacilli (b) which are continuous with similar elements in the pia mater (c); also the blood underneath the pia (d) is continuous with that outside the adventitia of the vessel (d'); the intima is broken through at (e) and a mural thrombus is indicated by (f), blood in the pia arachnoid spaces by (g), leptomeningeal artery (h), outer layer of ganglion cells (i), large ganglion cells (j); the mass of red blood cells (d") is continuous with that outside the adventitia (d'); Gram-Weigert stain; camera lucida.

On admission his temperature was 104.4 F., pulse 120, respirations 36. There was a marked soft swelling behind the right tonsil and of the soft palate, also an indurated swelling of the right side of the neck with a small pustule opening at the summit of the swelling. This swelling continued onto the right side of the chest. The pustule had a small necrotic center with a blue margin and a red zone about this. Bacilli were found in a smear from this pustule. The extremities were spastic. The pustule was excised and tracheotomy made. There were numerous small lacerations and bruises of the scalp and extremities from having fallen out of bed at home (convulsions?). The leukocyte count was 28,000 and the cerebrospinal fluid blood stained. He lived about 10 hours in the hospital, and died about 3 days after the onset. A diagnosis of anthrax was made.

Anatomic diagnosis (Dr. E. H. Hatton): Acute hemorrhagic leptomeningoencephalitis (anthrax infection); anthrax of the right side of the neck; edema of the glottis; acute hemorrhagic cervical lymphadenitis; acute anemia; acute hyperplasia (220 g.) of the spleen; recent tracheotomy; multiple small lacerations and bruises.

Brain.—The dura is everywhere tense and pale blue from the blood underneath it. When the dura is reflected, blood within the leptomeninges comes into view. This blood is thickest (2-6 mm.) at the frontal and parietal lobes. The convolutions of the top and base of the brain were only slightly visible shining through as gray opaque places. Wherever the tense arachnoid was torn in handling the brain, the surfaces of the convolutions were studded with petechial hemorrhages, 0.5 to 1 mm. in diameter, rather evenly distributed. On the cut surface of the cortex these hemorrhages were irregular, oblong, 1 to 2 mm. in diameter and limited almost entirely to the gray matter. The brain was extremely soft, almost jelly-like and weighed 1,470 gm. The cerebrospinal fluid was blood stained. The large arteries of the base of the brain were unchanged. A horizontal section through the basal ganglions made surfaces essentially similar to that shown in fig. 1. The hemorrhages were a little more numerous in the opercular and insular regions than elsewhere in the cortex but otherwise the distribution, size, shape and position (relative to the gray matter) were similar to brain 1; the white matter was similarly spared. The hemorrhages of the basal ganglions, though not quite so numerous as in brain 1, were similarly distributed. These hemorrhages were a little more numerous along the outer borders of the lenticular nuclei and along the vessels crossing the anterior limbs of the internal capsule. Such an arrangement was also present in brain 1.

Bacteriology.—In the stained smears of cerebrospinal, right and left pleural, and pericardial fluids and heart blood, large long-chain, square-end gram-positive bacilli were seen and pure cultures of similar bacilli were grown from each of these fluids. These were identified as anthrax, and pure cultures inoculated into guinea-pigs caused death within 20 hours. Hemolytic streptococci were also found in the pleural and pericardial fluids.

Microscopic Examination.—The changes in this brain differ from those in case 1 only in degree. There was more of a similar exudate in the pia mater, distending it to many times its normal width. The cerebral arterioles and venules had wide adventitia sheaths, the fibers of which were spread apart by a leukocytic bacillus-laden exudate and granular débris, stretching the wall to many times its normal width, the lumen either remaining normal with an intact intima or narrowed slightly by mural thrombi (fig. 3,e). When one of these vessels was followed along, an occasional break was found in its wall. At such a break there were red cells at the periphery of the already overdistended perivascular space and from here finger-like masses of red blood cells extended out into the brain substance (fig. 3c). There was continuity between the exudate in the pia and that in the adventitia sheaths of the cortical arteries and veins (fig. 2 a,b,c). Many of the adventitia sheaths were so widened that the surrounding brain substance was compressed, forming round or oval lightly-staining places, easily visible to the unaided eye when the stained section was held up to the light and as large as 0.5 mm. in diameter, when the normal diameter of the vessel would be about 0.02 mm. This increase in diameter was due primarily to changes in the adventitia. The few bacilli in the brain substance were either in the places of hemorrhage or in the bud-like extensions outward of the adventitia of some vessel (fig. 3a), in every case a continuity being easily established with some vessel. Only 3 bacilli were found in the vessel lumens during the careful study of several hundred sections. In no section were changes found in the intima except when associated with marked changes in the adventitia and media. Leukocytes were numerous in all these cerebral vessel lumens. The changes, other than already mentioned of the brain substance, were those of edema and acute inflammation, the increase in

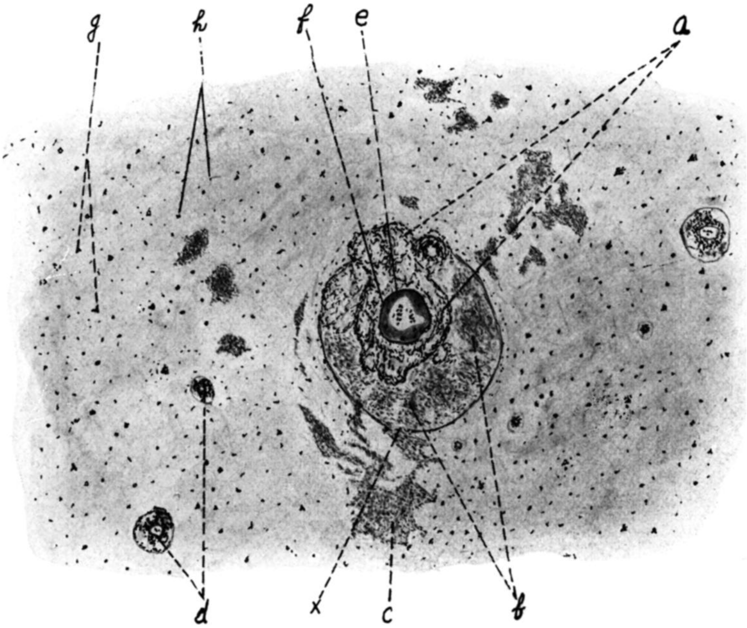

Fig. 3.—The cross section of an arteriole in the cortex of the left middle frontal gyrus 1 mm. in from the surface of the brain (case 2). The adventitia (a) is dissected widely from the media by edema and between it and the media there are many bacilli. There is a great deal of blood (b) outside the adventitia and this is continuous (x) with other masses of blood (c) still further out in the brain substance. In fig. 2 this blood about the vessel is but a part of the hemorrhage of the pia arachnoid and underneath the pia. This location of bacilli between the adventitia and media is also present in smaller vessels (d); a mural thrombus is indicated by (e), intima by (f), ganglion cells (g), glia cells (h). Gram-Weigert stain.

glia cells, satellitosis and shrinkage of the ganglion cells being most marked. All these changes became progressively less the deeper the section was taken.

Case 3.—L. K., a white man, aged 36 years, single, a laborer in a "curled hair" factory handling hair from South America, complained for the first time on the evening of May 1, 1920, of pain in the left side of the neck and left shoulder. May 2 (Sunday) he did not complain. May 3 he did not go to work; he complained some but helped his landlady about the housework. This history was obtained through an interpreter from the landlady, who could speak no English and appeared not to be very intelligent. On May 4 he drank some coffee and at 8:30 went to the toilet. He came out suffering from extreme dyspnea and collapsed in a chair. The dyspnea increased, he became more cyanotic and died at 9 a. m.; there was no attending physician and no diagnosis had been made.

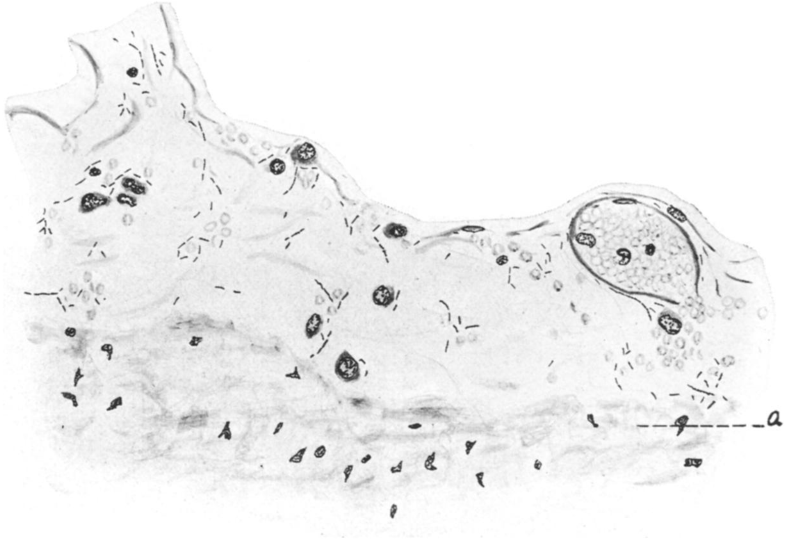

Fig. 4.—The pia with its fibers spread widely apart by edema (case 3). There are a few leukocytes and many bacilli in it; the cortex (a) underneath; gram-Weigert stain; Camera lucida.

Anatomic Diagnosis (Dr. J. P. Simonds).—Intestinal anthrax; anthrax septicemia; hemorrhagic leptomeningitis; anthrax edema of the scalp, neck and chest; hemorrhagic edema of the omentum, mesentery and tissues of the mediastinum; subpericardial hemorrhages; edema of the glottis; edema and hyperemia of the epiglottis; hemorrhages into the tissues of the mesentery; submucous gastric hemorrhages; hemorrhagic mesenteric lymphadenitis; acute splenitis; hydroperitoneum, thorax and pericardium; dilatation of the left ventricle of the heart.

Brain.—There was no injury of the outside of the skull. The pia arachnoid tissues of the vertex of the brain were filled with a bloody fluid which almost completely obscured the convolutions and was most abundant over the upper and lateral surfaces of the cerebrum, but present also though less abundant on the base of the brain and the cerebellum. The arteries at the base of the brain were collapsed completely.

Bacteriology.—In the stained smears from mesenteric lymph glands and spleen, large gram-positive square-end, rod-shaped bacilli in long chains and filaments were seen, in the former in enormous numbers, in the spleen relatively few. Cultures from the spleen and mesenteric lymph glands yielded bacilli similar morphologically to those seen in the smears, identified as Bacillus anthracis, and in the latter there were a few colonies of Bacillus coli as well.

The brain was received in formalin 3 hours after it had been removed, and it was still very soft. After a few days it was cut and no grossly demonstrable hemorrhages were found in its substance. Many of the vessels in the outer layers of the cortex and the striate vessels were distended with blood.

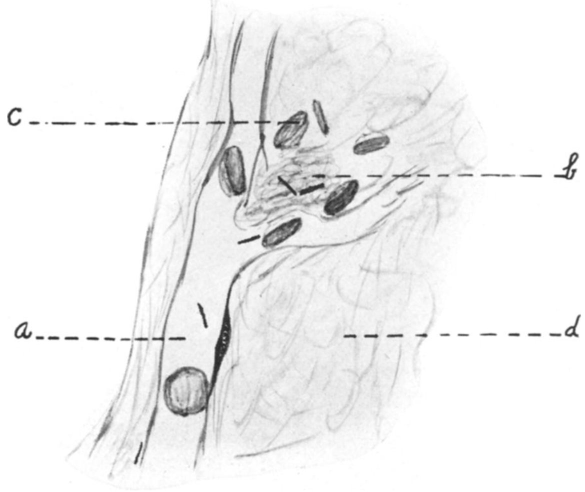

Fig. 5.—Early lesions in the adventitia of a leptomeningeal artery (case 3), lumen to the right, showing a vas vasorum (a) containing three red blood cells and two bacilli, a place of focal edema (b) with two bacilli in it, red blood cells (c) outside the lumen of the vas vasorum and (d) loose tissue of the adventitia; Gram-Weigert stain. X 700 diam.

Microscopic Examination.—In the spaces of the pia arachnoid there were large numbers of red blood cells and a few bacilli. In the meshwork of the pia arachnoid, besides these same elements, there were a few leukocytes, mostly mononuclears. The pia mater was a little thickened by a moderate amount of leukocytic exudate and bacilli (fig. 4) though to a less degree than in either of the other brains. The hemorrhagic leukocytic bacillus-laden exudate was continuous into the brain substance by way of the adventitia of the cerebral vessels in exactly the same way though to a less degree than in the other two brains. As in brains 1 and 2 the pia mater was in places dissected away from the brain by red blood cells which also partly filled the perivascular spaces of the vessels which pierced the pia. No thrombi were found in any of the cerebral or leptomeningeal vessels. There were numerous bacilli in the adventitia of the larger cerebral veins associated in places with patches of edema and dissection in some of which places the walls were entirely broken through. In the leptomengingeal vessels of small (less than 1/16 mm. in diameter) caliber there was no change except for an occasional bacillus in the adventitia.

One leptomeningeal artery, 0.5 mm. in diameter, was cut obliquely, almost longitudinally, and the vasa vasorum could be seen distinctly and traced into its media. There were numerous bacilli scattered along the walls of these tiny vessels and an occasional bacillus in the lumen (fig. 5a). More bacilli were counted in the lumen of one of these channels in one section than were found in the interior of all the larger vessels of the entire series. In the fork of the branch of one vas vasorum, outside of the channel, there was an irregularly oval, pink granular place about 20 mikrons in diameter containing 2 bacilli (see fig. 5b). There were 5 or 6 red blood cells outside this place in the delicate tissue of the adventitia (fig. 5c). These blood cells were also outside of the vessel channel. On account of the delicacy of the wall of this vas vasorum no definite discontinuity could be demonstrated. There were many other similar granular places of edema where the fibers of the adventitia of this and other vessels were widely separated. Some of the endothelial cells of the lining of the larger vasa vasorum were large and a few of them had swollen bacilli in their protoplasm. When a part of a chain of bacilli lay within and a part outside such a cell those within were swollen and those outside had their normal size and shape.

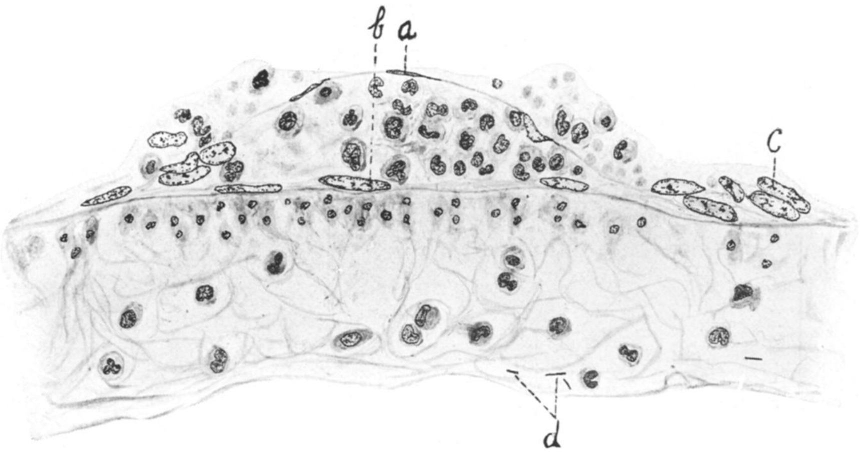

Fig. 6.—Subintimal infiltration of leucocytes; a, b and c, nuclei of endothelial cells; d, bacilli in a minute vas vasorum of the adventitia.

Careful search through the literature has yielded reports of 45 cases of anthrax with grossly demonstrable lesions in the brain: Bruce and Shennan [3] in their report of one case with leptomeningeal hemorrhage were able to collect 26 others. In addition to these Weichselbaum[4] reported 2 cases; Burmeister,[5] 2; Meyer,[6] 1; Wepfer,[7] 2; Wagner,[8] 1; Reece,[9] 1, and Greenfield,[10] 3. Reports of other cases are referred to elsewhere in the text. Of these 45, sufficient details are given in 29 to note the following: average duration of illness, 2 or 3 days; malignant pustule of the head or neck, 17; intestinal and pulmonary, 7; malignant pustule of the extremities, 3; edema of the neck, 2; hemorrhage into the meninges, 20; hemorrhage into the meninges and brain substance, 9. In only one of the 7 intestinal and pulmonary cases did hemorrhages occur in the brain substance.

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[3] Rev. Neurol. and Psychiat., 1910, 7, p. 521.

In every reported case in which the details are given there were symptoms of cerebral irritation coming on usually several hours before death, and sometimes even 1 to 5 hours before. The great majority of deaths were ascribed to the cerebral lesions, whereas in those with delayed cerebral symptoms death was invariably said to be due to other lesions, notably edema of the larynx. In any event, however, the minds of those with cerebral lesions do not remain clear up to the time of death and it was even suggested by Bell[11] that whenever symptoms of cerebral irritation appear in the course of an anthrax infection, it is most probably due to brain lesions. There are several cases recorded with the first symptoms meningitic, a rather sudden onset with general malaise, headache and often dizziness. Nausea, vomiting, chilliness, pains in the back and extremities and restlessness soon follow. In less than 24 hours the patient may become delirious. This is usually accompanied by convulsions, which may be so violent as to require restraint; then coma and death. Difficulty in swallowing and breathing may appear early and occurs most commonly when there is a malignant pustule or edema of the neck. Muscular twitchings may be present very early. The fever is high at first, 100 to 105 F., later falling to 99 degrees or even lower. Leukocytosis is the rule and may be as high as 75,000 per cu mm. When spinal puncture was made blood tinted fluid was obtained, which was loaded with bacilli. There is one recent report[11] of a recovery from anthracic meningitis where the diagnosis was made by spinal fluid examination. These spinal fluid findings are so constant that in every case in which blood stained fluid is obtained smears should be examined immediately and if large bacilli are found the patient isolated until the organism is carefully identified. This identification cannot be too thoroughly carried out, as is shown in one of the cases of this series in which bacilli were found, grown on plates, and discarded as B. subtilis.

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[4] Internat. Klin., Rundschau., 1888, 2, p. 1369.

[5] Inaug. Dissertat., Rostock, 1907.

[6] Deutsch. med. Wchnschr., 1901, 40, p. 1027.

[7] Inaug. Dissertat., Heidelberg, 1910.

[8] Archiv. der Heilkund, 1874, 15, p. 1.

[9] Lancet, 1917, 1, p. 409.

[10] Local Gov. Bd., Med. Officer Reports, London, 1881.

[11] Czyhlarz, Ernst: Wien. klin. Wchnschr., 1916, 29, p. 768.

There are two routes by which anthrax bacilli may invade the brain and its membrane, the lymphogenous and hematogenous. Risel,[12] Bruce and Shennan,[3] Fulci,[13] and Herzog[14] have reported cases that seem to show that anthrax bacilli may reach the brain through the lymphatics. There is no such direct evidence that anthrax bacilli do localize in the brain by way of the blood stream, though it would be extremely difficult to explain such localizations in any other way in those cases in which the only other focus in the body is in the intestine or lungs or on one of the extremities. Of the 36 cases in which the location of the primary lesion was stated, 17 belong in this last category. Although there may be some definite relation between the location of the primary lesion and brain complications, such a relation is not borne out by the statistics.

Not only from the study here presented but also from the studies of similar lesions by others I am impressed with the importance of recognizing two gross anatomic pictures produced in the brain by anthrax; one, the soft red viscid brain with blood only in the leptomeninges, the other, the soft red viscid brain with multiple hemorrhages of characteristic shape and distribution in its substance. Other observers have failed to emphasize these changes.

The brains of this report are excellent examples of these two types (brain 3, type 1; brains 1 and 2, type 2). It is fair to state for the present that no other disease is known to produce such changes as in type 2, namely, hemorrhages with such characteristic shape and distribution; the alterations are so marked and particular as to be characteristic of anthrax and in this sense useful in the recognition of anthrax by postmortem examinations of the bodies of persons the circumstances of whose deaths are unknown.

The microscopic picture is even more characteristic and Wagner,[8] Fraenkel,[15] Risel,[12] Symmers and Wilson,[16] Fulci,[13] and Herzog,[14] in reporting cases have fairly adequately described these changes.

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[12] Ztschr. f. Hyg. u. Infektionskr., 1903, 42, p. 380.

[13] Histol. y. Histopath. Arb. u. d. Grosshirnrinde, 1913-14, 6, p. 161.

[14] Beitr. z. path. Anat. u. z. allg Path, 1915, 60, p. 513.

[15] Ztschr. für Hyg. u. Infektionskr., 1898, 27, p. 315.

[16] Jour. Path. and Bacteriol., 1908-09, 13, p. 251.

The most comprehensive work was done by Fulci on a brain with cortical hemorrhages and Herzog on three brains without cortical hemorrhages, both of whom concluded that the vascular lesions were produced by agents acting outside of the vessel lumen. Neither of them found bacilli in the vessel lumens. Such changes as occur early in the walls of the leptomeningeal vessels were not described by any of these investigators.

The essential microscopic picture is one of acute inflammation plus hemorrhage. The large amount of hemorrhagic exudate and almost complete absence of retrogressive changes is in strict accord with anthrax lesions elsewhere in the body. The large vessels of the leptomeninges seem to be the first to show this change and of these the veins are affected most. The changes in brain 3 are evidently of shorter duration than those in brains 1 and 2 on account of the relatively larger numbers of red blood cells, small numbers of leukocytes and bacilli in the exudate, the small degree of extension of this exudate into the brain substance along the cortical vessels and the relatively small amount of edema in the walls of the cerebral vessels, especially the cortical. In brain 2 the inflammation is probably of longer duration than in either brain 1 or 3, because the leukocytes, fibrin, and bacilli of the exudate predominate; there has been more and deeper extension into the brain substance along the vessels and there is much more thrombosis of the cortical vessels. The changes in brain 1 are also of considerable duration, but the exudate around the cortical vessels is much more hemorrhagic and there is relatively little thrombosis. These differences in the inflammatory process of the brain are in strict accord so far as one can judge with the duration of brain involvement; although in the third case no account of cerebral symptoms was obtained.

The amount of cortical changes seems to depend somewhat, therefore, on the duration of the inflammatory process and the extension of the exudate from the leptomeninges into the brain substance along the vessels; this I believe explains why in some of these cases there are no intracerebral hemorrhages. Such an extension inward of the exudate, especially of the red blood cells, is facilitated in the case of the striate vessels by the large channels in which these vessels lie.

It must be remembered that the microscopic study of these brains was necessarily limited to relatively small portions and that if similar studies could be made of all parts we might possibly come nearer to the truth. But so far as one can judge with the unaided eye and by taking sections not in series from other regions, the changes described are representative of the whole.

SUMMARY AND CONCLUSIONS

A number of cases of anthrax have occurred in Chicago recently, many of them among workers in the "curled hair" industry. In at least three of these cases there were brain complications and in two of the three anthrax was not suspected until after death. It seems reasonable therefore, to suspect that from time to time similar cases may go unrecognized. In an effort to eliminate such a possibility a careful microscopic and bacteriologic study should be made of every bloody spinal fluid, provided one is certain that the blood is not a contamination; and especially should this be done if there are symptoms of cerebral irritation.

These cases can be easily recognized at necropsy by the soft, red viscid brain, especially if there is a history of an acute illness not resulting from trauma; and it must be remembered that there may be one of at least two types of gross changes—either the brain with the characteristic intracerebral hemorrhages or the brain with blood only in the membranes. On account of the large quantity of blood which is sometimes present it would be easy for one who had not seen such brains to attribute the cause to trauma, but this may be avoided by recognizing the inflammatory quality of the changes and by checking up with stained smears of the exudate.

The lesions in the brain in anthrax are essentially extravascular, meningo-encephalitic, and the acute hemorrhagic nonpurulent nature of the inflammation with destruction of tissue by edema is similar to that produced by anthrax in other places of the body. With suitable stains large numbers of bacilli may be found in these lesions.

The infection of one of the brains of this series (Case 3) was evidently by the hematogenous route, first, because the primary focus was so far removed (intestine) and second, because of the finding of small bacillus-containing foci of edema and dissection along the vasa vasorum of the leptomeningeal vessels.